

*The Author's Compts*  
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# Intra-Venous Injections in Blackwater Fever.

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By  
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L.R.C.P., Late Physician to King Edward VII.'s Hospital for  
Officers, etc.



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I AM sure all who practise tropical medicine will welcome a suggestion, which may prove of assistance in the treatment of this disease. In the case reported below, I adopted a method of introducing fluid, which is, I believe, original in these cases, for I can find no mention of it in the treatment advised in text-books on the subject.

Major H., aged 35, 5 ft. 7 in., 9 st. 3 lb. in clothes, arrived from Southern Nigeria on June 24th. I saw and examined him on June 26th, as he complained of muscular rheumatism and of being out of sorts. He had led a very strenuous life in Africa, had suffered from enteric and dysentery, and had had three or four attacks of malaria, but none for the last three years. There was no enlargement of the liver and spleen. I sent him to Aix-les-Bains to have a course of treatment for rheumatism, hoping the change would do him good. On his return from Aix he went on a round of visits.

On the evening of November 7th, while on a visit in North Wales, he felt "shivery" and took 10 grains of quinine; less than two hours later, when passing urine, he noticed it was black, and as he passed a normal amount, obviously most of this urine had been secreted before the quinine was taken. The patient realized at once what was wrong, and on November 8th he motored 18 miles to Llandudno and consulted Dr. Bold Williams, who advised him to go to London to King Edward VII.'s Hospital for Officers at once, in preference to motoring back to his friends. He arrived in London late at night, and was admitted to King Edward VII.'s Hospital for Officers. He had been violently sick several times on the journey, and was very exhausted.

I ordered a warm saline rectal injection, and a hypodermic of  $\frac{1}{4}$  grain of morphia. He had a restless night, and two rigors in the early hours of the morning of the 9th, one at 3 a.m. and one at 6 a.m. When I saw him at 10 a.m. on the 9th, his condition was very grave. The vomiting was more or less continuous, and of a green colour, the pulse was poor, and the vessels somewhat empty. The patient was jaundiced, and the urine was quite black and contained a small quantity of albumen. As it was obviously impossible to give fluid by the mouth, and I wished to avoid the absorption of toxins from the bowel, I decided to inject normal saline, at the temperature of the patient, direct into the vein. I injected 25 oz. of saline by means of the needle now



should not be given by the mouth, as even a spoonful of fluid brought on vomiting. Later in the day, I ordered  $\frac{1}{2}$  grain of calomel every hour for three doses and two hours after the last, a dose of Rubinat was given but not retained; the bowels, however, were opened three times, the motions being very offensive. At 7 p.m. I injected 25 oz. of normal saline into the vein, and gave  $\frac{1}{4}$  grain of morphia late at night. It was necessary, in view of his restlessness, to repeat this dose of morphia at 3 a.m.

At 10 a.m. on the 10th, I gave 25 oz. more fluid into the vein, and, as his vessels were now quite decently full, I did not consider it necessary to repeat the injection at night. During the early hours of the morning of the 11th, the patient's condition became somewhat collapsed, and Dr. Harold R. Moxon, the house surgeon, gave a rectal saline of one pint, following with  $\frac{1}{4}$  grain of morphia. It was necessary to repeat this saline at 7 a.m. At 10 a.m., I repeated the intra-venous injection, giving another 25 oz., so that altogether from the beginning, I injected into the veins five pints of normal saline, temperature about  $103^{\circ}$ .

The bowels having been opened and washed out with glyco-thymolin and water, and the stools being far less offensive, there was no reason why normal saline should not now be given into the bowel. This was done every six hours for the next twenty-four hours, as much as he could retain; glucose and bi-carbonate of soda were added to these salines. During all this time the urine steadily improved in colour and there was no albumen after the second day. There were murmurs to be heard in the heart at all the orifices, and pulsation at the neck was very marked indeed. It was necessary on several occasions to give a hypodermic of strychnine and digitalin. The patient was allowed to sip fluid as soon as the sickness had stopped, and the rectal injections of glucose were given twice a day for three days more. From this on he made uninterrupted progress, the temperature became sub-normal, and the murmurs gradually disappeared, but he was kept in bed for three weeks, until all danger of renal complication had passed.

The analysis of urine on admission and the condition of of the blood were:—

## URINE.

*November 10th.*

*Spectroscopic Examination:—*

The urine shows the bands of methæmoglobin very clearly.

## Analysis:—

Acid	-	-	-	Sp. Gr. 1014.
Albumen	-	-	-	Abundant.
Sugar	-	-	-	None.
Chlorides	}	-	-	Normal.
Sulphates				
Phosphates				
Urea	-	-	-	2.9 per cent.



*Microscopical Examination of Deposit :—*

Some granular casts and vesical epithelial cells, together with small numbers of pus corpuscles, are present. I find no red blood cells, parasitic ova, or shreds of growth.

*Quantitative Estimation of Albumen.*

Albumen - - - - - '04 per cent.

*Bacteriological Examination :—*

The urine contains an abundant growth of *B. coli*, but no other pathological organisms are present.

## BLOOD.

*Microscopical Examination :—*

Red cells stain fairly well. There are a few poikilocytes, and there is some polychromatophilia. Some corpuscles are hypertrophied, and exhibit basophilic degeneration. There is no suggestion of the presence of intra-corpuscular malarial parasites, and though some pigmented polymorphonuclear cells are present, I find no gametes or other indication of present malarial fever.

*Differential Count of Leucocytes :—*

Polymorphonuclear cells	-	-	-	-	67·0
Lymphocytes	-	-	-	-	30·0
Eosinophil cells	-	-	-	-	·5
Mast cells	-	-	-	-	·5
Transitional cells	-	-	-	-	·25
Mononuclear cells	-	-	-	-	1·75
Myelocytes	-	-	-	-	·0
					<hr/>
					100·0

*November 13th.—Estimation of Red and White Corpuscles.*

Red cells	-	-	-	-	1,400,000 per c.mm.
White cells	-	-	-	-	12,300 „
Hæmoglobin (Haldane)	-	-	-	-	27 per cent.

*November 20th.—Estimation of Red and White Corpuscles.*

Red count	-	-	-	-	2,140,000 per c.mm.
White count	-	-	-	-	4,700 „
Hæmoglobin	-	-	-	-	49 per cent.

(Observations taken before breakfast.)



*November 27th.—Estimation of Red and White Cells.*

Red cells	-	-	-	-	3,420,000 per c.mm.
White cells	-	-	-	-	7,900 „
Hæmoglobin	-	-	-	-	77 per cent.

For the last fourteen days the urine was quite normal.

I obtained from the patient the names of the places he had visited in Southern Nigeria, where there had been cases of blackwater fever, and his report was as follows:—

District visited by me.	Year.	No. of cases of blackwater fever to my knowledge.
*Ibadan-Oyo -	1906 -	3
*Ibadan-Oyo -	April and May 1913 -	1
Ogoja -	1911 -	2
Ogoja -	September to November 1912 -	Nil to my knowledge.

\* Between 1906 and 1913 I was not in this district.

I recollect being bitten on several occasions by tsetse fly in the Ibadan-Oyo district, where these flies are numerous, especially north-west of Oyo. Many tsetse are also found in the Ogoto district.

(Signed) A. H. W. H.

Major H. always slept under a mosquito net at night, and is positive he was not bitten by mosquitos, but while big game shooting during the daytime, in May, he was bitten by tsetse fly. There may be some infection carried by this fly causing blackwater fever, for I feel convinced that in this case malaria will not explain the attack. He had been free for three years, and the blood not only failed to show the malarial parasite, but there was also an absence of the large mononuclear increase met with in chronic malaria. I think it would be of interest, and might throw light on the origin of blackwater fever, if those who come across this disease would ask the patients, whether the tsetse fly had been abundant in the area in which they had been a few months before the onset of the attack.

In conclusion, while realizing to the full the help of the exceptional care given to Major H. by Dr. H. R. Moxon, Sister Agnes, and the nurses of King Edward VII.'s Hospital for Officers, I feel convinced that but for the intravenous injections he would have died.

I am indebted to Dr. Harold Spitta for the reports on the urine and the blood.





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